CASE REPORTS

- **◀** Renocolic Fistula
- ◀ Meningovascular Myelitis in Early Syphilis

Renocolic Fistula

H. VERRILL FINDLAY, M.D., Santa Barbara

ENOCOLIC fistula was reputedly described by Hippocrates. Rayer¹⁰ in 1841 gave a clear description of this lesion and presumed that it followed a long standing renal infection with probable perirenal abscess. No better theory of the production of this lesion has been produced since Rayer's time.

In spite of the long history of our knowledge of this lesion reports of cases are relatively rare. Mertz,⁵ in 1931, collected from the literature reports of numerous cases of upper urinary tract fistula to show the relative frequency of occurrence at various sites. He found reports of two renogastric, three renoduodenal, nine renobronchial, and 29 renocolic and ureterocolic fistulae. By way of contrast and to show the greater prevalence of lower urinary tract fistula, it may be noted that Fowler² in 1928 reported 500 cases of vesicointestinal fistula. Vermooten and McKeown,12 reviewing the literature in 1933, found reports of 26 cases of renocolic fistula. Ratliff and Barnes' in a review in 1939 found 11 more. Pedroso, Anglada and Pedroso⁸ in 1940 reported an accumulated total of 43 cases, including two of their own. A cursory review of the literature since then shows Markowitz and Katz4 have reported two cases, Minuzzi and Torresi7 one, and Miller one and Liebich one. This paper adds one more, making a total of 49 cases reported to date.

As its name implies, a renocolic fistula is one connecting a kidney and the colon. Rayer was of the opinion that the lesion was primarily the result of chronic renal inflammation which produced a perirenal abscess that ultimately ruptured and drained into the colon.

Study of the reports of individual cases leads to the conclusion that this is the way most of these fistulae are produced. The lesion is always primary in the kidney. No case has been recorded with an initial bowel lesion. As the colon at the region of the hepatic and splenic flexures is in intimate contact with the anterior surface of either the right or left kidney, rupture of a perirenal abscess into the colon is understandable. A surprising phenomenon is that the fistula passes through the wall of the cortex rather than the wall of the pelvis of the kidney.

Ratliff and Barnes, in reviewing reports of 37 cases, noted the following associated and probable causative lesions: renal tuberculosis in five cases, renal calculi in 14, and pyelonephritis in 18. No case in which there was associated malignant growth in the kidney was found by them, and they found no case in which the primary lesion was in the bowel.

The patient's history and results of urinalysis may suggest renocolic fistula, but diagnosis may be confirmed only by retrograde pyelograms. A patient who has this disease usually appears debilitated and is chronically ill. Frequently there is a history typical of perirenal abscess, with symp-

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toms suddenly subsiding but blood-streaked stools appearing concomitantly. There are not, in the typical case, constant symptoms of bowel disorder.

Findings upon physical examination may not be characteristic or striking except for evidence of chronic sepsis. Urinalysis usually discloses marked pyuria. Intravenous pyelograms do not constantly nor reliably demonstrate the lesion. Cystoscopic study reveals chronic cystitis. A pyelogram of the kidney, if the kidney is adequately filled, will demonstrate the fistulous tract, and the media should flow into the colon after occasionally filling a perinephric abscess cavity. Typically, the affected kidney pelvis cannot be distended to the point of pain because the injected media will escape into the colon. A barium enema is unnecessary for diagnosis and usually of little help. Riaboff and Feldman demonstrated the fistula by means of a barium enema in one case.

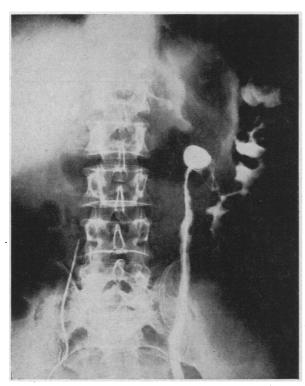
The curative treatment of renocolic fistula is nephrectomy with ligation of the fistulous tract as near to the colon as possible. In aged, debilitated or very ill patients this may not be feasible. Ratliff and Barnes report an operative mortality of 33 per cent in cases reported to 1939. A perinephritic abscess may be drained primarily and later a nephrectomy and ligation of the fistula done. An occasional patient will withstand only nephrostomy. Some die without benefit of operation. In some cases colonic cutaneous fistulae occur postoperatively. However, this lesion is so severe in effect upon the patient's health that considerable operative risk is warranted.

CASE REPORT

A 56-year-old white male consulted his physician because of pain in the left renal area, dysuria and cloudy urine for over three months. In 1938 the patient had been operated upon for removal of a left renal calculus following several disabling attacks of colic, but he had been told by the surgeon that the stone had not been removed. The patient was positive that there had been no diagnosis of perirenal abscess preoperatively, and questioning elicited no recital of symptoms at that time which might have indicated the presence of the lesion. Convalescence from the operation was slow, with urinary drainage from the wound for eight weeks. After recovering, the patient resumed work as a rancher and was free of symptoms for five years.

Two or three years before the present illness, aching developed in the left flank and recurred periodically. However, there were no urinary symptoms. Three months before he was examined by the author, the patient had had what he termed "flu" which began as an upper respiratory infection and then "settled in my bowels." There was left upper abdominal pain and aching and tenderness for about two weeks, followed by loose stools and disappearance of these symptoms. Blood was not observed in the stools at this time, although it was not searched for.

Weak, emaciated, discouraged, chronically ill when he came under the author's observation, the patient was hospitalized without delay. General examination disclosed nothing of note. The abdomen was thin and no masses were



After rapid injection of 50 cc. sodium iodide solution, the tiny pelvis surrounding the left renal calculus, the fistulous tracts, and partial filling of the descending colon were demonstrated. This film established the diagnosis.

found, although there was some tenderness over the anterior end of the well-healed left lumbar scar. External genitalia were normal and the prostate normal in size and texture for his age. A voided specimen of urine was cloudy, and over 1.0 cm. of pus settled in the bottom of the centrifuge tube. Results of urinalysis were otherwise normal. Erythrocytes numbered 4.6 million with a hemoglobin value of 86 per cent and leukocytes numbered 6,100. The nonprotein nitrogen level was 37.5. Results of a Kahn test were normal. Urine specimens from the left kidney and bladder contained much pus and B. coli on culture, whereas urine from the right kidney contained no pus and no growth on culture. A roentgenogram of the chest showed no abnormality of the heart or aorta. There was moderate generalized emphysema.

A cystoscopic study showed no intrusion by the prostate, but generalized subacute cystitis was noted. Urine came from the right ureteral orifice in spurts, but none was seen coming from the left orifice. A No. 6 (French) catheter was passed up each ureter easily and a normal flow of clear urine was obtained from the right kidney, but only a small amount of thick pus could be aspirated from the left. Roentgen studies, including pyelograms, were made and a few calcific flecks were noted in the parenchyma of the right kidney, which was otherwise normal. At the upper end of the left ureteral catheter was a round, laminated calculus 2.5 cm. in diameter, apparently in the left kidney pelvis. When a small amount of 121/2 per cent sodium iodide solution was injected into the pelvis through the catheter, a tiny pelvis closely surrounding the calculus was demonstrated with a satisfactory filling of the ureter. When larger amounts of the solution were injected, the three sinus tracts leading downward and lateralward from the kidney pelvis were filled and eventually the descending colon was partially filled. As the right kidney was found to be functionally good although probably containing early calculi, operation was decided upon to close the fistula and eliminate the serious urinary tract infection.

Left nephrectomy was done with the patient under pentothal and gas-oxygen anesthesia. The left lumbar scar of the previous operation was excised and the left lumbar muscles incised. As the normal perirenal fat had been replaced by dense scar tissue, dissection was extremely difficult. Although the left renal calculus was felt, no surrounding tissue suggestive of a kidney was palpated. With the field properly exposed, sharp dissection was used and a small amount of fibrotic, fat-surrounded tissue enclosing the calculus was freed with an adjacent 12 cm. of ureter. The fibrotic band, thought to be the fistulous tract, was then carefully followed downward and medialward through a mass of fat until the wall of the colon was exposed. This band was doubly ligated with a No. 00 chromic tie. Within the renal pedicle was a minute artery which was ligated and divided. Then the ureter was similarly treated and the mass removed. The wound was closed in layers with a drain after 4 gm. of sulfanilamide powder had been dusted into it. Time for the procedure was 45 minutes, and the patient was taken from the operating room in good condition.

Convalescence was stormy. The patient reacted poorly and slowly from anesthesia, showing profound narcosis and marked hypotension. A transfusion of whole blood was given, whereupon the temperature rose to 107° F., although a recheck of the donor's and the patient's blood revealed no incompatibility. After three plasma transfusions and other supportive measures in the first 48 postoperative hours, progress, although slow, was satisfactory and the patient was out of bed in seven days. Weakness and anorexia continued, but there were no specific complaints. On the ninth day following operation, a severe left corneal ulcer, which was refractory to treatment, developed. Penicillin was given for ten days postoperatively and the operative wound healed satisfactorily. When the patient left the hospital 17 days after operation there was a small amount of serous wound drainage. Few pus cells were found in an examination of the urine at that time.

The pathologist reported that the mass of fat and fibrotic tissue removed weighed 55 gm. without the stone which measured 2.7×1.7 cm. The only tissue which could possibly be identified microscopically as kidney consisted of a few areas of dilated renal tubules and renal pelvis epithelium. The rest of the areas showed only fat, chronic inflammatory tissue, and fibrosis.

The patient made a slow but progressive recovery. A small amount of clear serum continued to drain from the wound for four months before healing was complete. At no time did the draining serum look or smell like urine or fecal material. The urine remained clear except for an occasional leukocyte. When last seen, six months after operation, the patient was doing light ranch work and the wound had healed. There were still a few leukocytes in the urine.

COMMENT

In reviewing this case, doubt arises as to when the renocolic fistula developed. It may have developed soon after the initial operation, or it may have occurred when pain in the left loin was noted five years later. However, it would seem most likely that it developed during the bout of so-called "flu," some three months prior to the second operation, for not until then had the patient had severe pain and tenderness in the left renal area, suggesting an abscess, and it was not until then that urinary symptoms were noted.

CONCLUSIONS

Renocolic fistula is not a commonly reported lesion. Once suspected, diagnosis of it can be certainly established by

retrograde pyelogram. The treatment of choice is nephrectomy with ligation of the fistulous tract.

A case is reported in which recovery followed operation. 1515 State Street.

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Meningovascular Myelitis in Early Syphilis

ERNEST K. STRATTON, M.D., San Francisco

ACUTE myelitis of syphilitic origin is a rare condition in the United States if the available case reports are a true indication of its frequency. For it to occur within six months following the primary infection, however, is still more unusual.^{2, 3, 6} Many of the cases of acute myelitis reported during this period have been due either to the Jarisch-Herxheimer reaction following the first injections of arsphenamine or to a hemorrhagic myelitis from arsenical intoxication.^{1, 4, 7}

When this tragic involvement occurs during therapy a differential diagnosis between these three possible causes is in many instances a difficult one to make. The neurological signs and symptoms of cord damage resulting from any one of them are more or less the same, regardless of the precipitating cause. If the patient recovers, credit is usually given to the method used in handling of the case and an etiological diagnosis is established on that basis. For the patient who dies, only examination of a cord section absolutely ascertains which process is the responsible one.

The following case report illustrates the problem involved under such circumstances:

CASE REPORT

The patient, a white female, was examined March 8, 1943. The skin on the entire body was covered with maculo-papular eruptions of secondary syphilis and there were mucous patches in the mouth, as well as generalized lymphadenopathy. The patient complained of dull bifrontal headache. Results of blood tests by both the Kolmer and Kahn methods were four plus. Neoarsphenamine, 0.9 gm. intra-

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venously, was given every fifth day for eight doses. Following the eighth injection, generalized arsenical dermatitis appeared. The patient was very ill, the skin of the entire body, including hair and nails, exfoliated, and there was a decrease in body weight of 40 pounds to a total of 115. After eight weeks during which vitamin therapy was the only medication, the patient began to improve and regained 20 pounds. A blood test on June 25 showed that the Kolmer reaction had been reduced to negative and the Kahn to three plus.

Around the first of September, however, the patient began complaining of intermittent deep aching of both lower extremities from the knees down, mostly when sitting. It was present day and night. Also, she complained of a progressive weakness of the knees and a fear of falling. There was incoordination of the legs in walking and she began staggering. The incoordination was worse on descending stairs but present also in ascending. There was "numbness" from the hips down, with decreased perception of touch and temperature. The patient was unable to void except with considerable effort and straining. Marked constipation was present.

A spinal fluid examination made at this time showed 83 cells per cu. mm.; a positive globulin; total protein was 0.107 mg. per 100 cc.; colloidal gold 1113331110, and Kolmer reaction two plus.

The patient had received no antiluetic treatment of any kind since April 20, the date the arsenical toxic skin symptoms first appeared, but in view of the positive luetic findings in the spinal fluid as well as the signs of progressive cord damage, specific treatment was again begun, this time starting with bismuth subsalicylate intramuscularly (0.13 gm.). After two months of bismuth therapy, no improvement was observed, so on November 11 1 gm. of tryparsamide was given intravenously. Nausea followed this injection but no skin reaction. Two grams more of tryparsamide was given on each of the following dates: November 18 and 26 and December 2. Neurological symptoms became more pronounced. The patient complained almost constantly of the sensation of a tight band or girdle squeezing the abdomen. The legs became progressively weaker, the patient fell to the floor and was too weak to arise without assistance.

Physical examination: The general physical examination revealed nothing unusual. The blood pressure, blood count and urinalysis were essentially normal.

Neurological examination* revealed an uncertain gait, positive Romberg, moderate general wasting of the muscles and marked ataxia. Sensation to touch about the nose was diminished; that from T 10 was variable, with saddle anesthesia. Vibratory sensation was absent below the knees. Reflexes in the upper extremities were exaggerated bilaterally as were the patellar jerks, but the ankle jerks were absent. Plantar responses were equivocal. Examination of the cranial nerves gave essentially normal results.

More tryparsamide was given, followed by a gain in weight and general improvement. However, the bladder, bowel and leg paralysis became more complete. After eight weeks, cystitis, pyelitis and pyelonephritis developed rapidly, and death resulted from streptococcal septicemia and bronchopneumonia in February, 1944. A spinal fluid examination made a few days before death showed the Kolmer to be negative in all dilutions, and the Kahn two plus, the cell count 74 and the colloidal gold 0012332100.

Microscopic findings* in the spinal cord were reported as

^{*}The neurological examination and report and the pathological examination and report were made, respectively, by Dr. Helen Starbuck, San Francisco, and Dr. Melvin Friedman, University of California Medical School, San Francisco.